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Emergence of serranid pigment abnormality syndrome (SPAS) in wire netting cod (*Epinephelus quoyanus*) from Heron Island on the southern Great Barrier Reef

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Abstract. Coral reefs worldwide are under increasing stress from anthropogenic impacts, but there are relatively few reports of increased rates of disease in coral reef fish. Herein we report the emergence of abnormal skin lesions in wild-caught wire netting cod (*Epinephelus quoyanus*) near Heron Island in the southern Great Barrier Reef. The lesion involves conspicuous darkening and disorganisation of the brown 'wire netting' colouration pattern typical of this species, most commonly on the lower jaw, premaxilla and head, with occasional involvement of the flanks and dorsal fin in some fish. The lesion was not present during research conducted in the mid-1990s; however, since it was first recorded in 2012, the prevalence of grossly visible lesions has increased to 16.9% in 2017, with fish >340 mm long most affected (prevalence 64.7%). These data suggest emergence of the lesion is a recent phenomenon and that causative factors may be age related. Abnormal pigmentation lesions have only been observed to affect *E. quoyanus* and coral trout (*Plectropomus leopardus*; since 2010). Given the species affected and the currently unknown aetiology of these lesions, we name the condition serranid pigment abnormality syndrome (SPAS). Further research is required to determine its geographic distribution, establish causation and describe the course of disease in *E. quoyanus*.

Additional keywords: chromatophoroma, coral reefs, disease, health, Serranidae, teleost.

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Introduction

Baselines of 'normal' disease in wild fish populations remain elusive (Lafferty *et al.* 2004; Ward and Lafferty 2004), although it is often thought that emergence of new diseases may be an indicator of environmental stress (Harvell *et al.* 1999; Lafferty *et al.* 2004). It is well documented that coral reefs worldwide are under increased stress due to global warming (Couch *et al.* 2017; Hughes *et al.* 2017) and a range of anthropogenic stressors, including terrestrial run-off (McCulloch *et al.* 2003; Brodie *et al.* 2012; Wenger *et al.* 2016), overfishing (Cinner *et al.* 2009; Lamb *et al.* 2015) and tourism (Juhasz *et al.* 2010; Lamb and Willis 2011; Au *et al.* 2014). On the Great Barrier Reef (GBR), these stressors have mostly been documented to affect corals in the form of bleaching events (Hughes *et al.* 2017), outbreaks of crown of thorns starfish (Fabricius *et al.* 2010; Wooldridge *et al.* 2015) and increased prevalence of coral diseases (Chen *et al.* 2017; Pollock *et al.* 2017).

In contrast, relatively few reports of disease outbreaks in wild fish or shellfish are found in the scientific literature on coral reefs. In the Caribbean, an acute large-scale epizootic in sea urchins (*Diadema antillarum*; see Lessios *et al.* 1984; Lessios 1988) preceded phase shifts towards algal dominance in coral reefs in the early 1980s (Idjadi *et al.* 2010). Although a disease agent was never identified as the cause of the initial mortality event, a microbial pathogen was suspected (Bauer and Agerter 1987) and it has taken nearly 30 years before sea urchin populations (and corals) in the affected regions began to recover (Idjadi *et al.* 2010; Rodríguez-Barreras *et al.* 2015). Then, in 2000, spiny lobsters (*Panulirus argus*) in the Caribbean were found to be infected with a novel virus (*Panulirus argus* virus 1 (PaV1)) that caused disease and mortalities in juvenile lobsters (Behringer *et al.* 2011).

In teleosts, common naturally occurring parasites can occasionally cause disease in wild coral reef fish under certain conditions (Bunkley-Williams and Williams 1994; Landsberg 1995), whereas epizootics caused by bacterial disease agents, particularly *Streptococcus iniae* and *Streptococcus agalactiae*, have been reported in several countries (Ferguson *et al.* 2000; Bowater *et al.* 2012; Keirstead *et al.* 2014). Some populations of coral reef finfish near Hawaii have been described with skin diseases in the form of chromatophoromas (including melanomas) with unknown aetiology in butterflyfish (Okihiro 1988) and surgeonfish (Work and Aeby 2014), whereas bicolor damselfish (*Stegastes partitus*) from Florida are affected by an

Table 1. Serranid pigment abnormality syndrome (SPAS) prevalence and other data from field sampling of *Epinephelus quoyanus* from Heron Island between May 1996 and October 2017

Water temperature data are given as the mean ± range for individual years and as the range for the period 2010–17. TL, total length

Year	Month	Water temperature (°C)	Number of fish sampled	Mean TL (mm)	TL range (mm)	Number of fish ≥340 mm TL sampled	SPAS prevalence (%)	SPAS prevalence in fish ≥340 mm TL (%)
1996	May	23 ± 1	158	295.4	230–370	19	0	0
2010	Nov.	25 ± 1	6	321.7	295–360	2	0	0
2011	Oct.	24 ± 1	11	301.3	270–360	2	0	0
2012	Oct.–Nov.	24 ± 1	43	306.4	172–370	11	2.33	9.1
2016	Oct.	24 ± 1	64	284.2	145–355	6	6.25	50
2017	Oct.	25 ± 1	65	306.8	210–380	17	16.92	64.7
2010–17	Oct.–Nov.	24–25	189	298.6	145–380	38	8.46	42.1

infectious viral disease (neurofibromatosis) involving development of neurofibromas and chromatophoromas (Schmale *et al.* 2002).

Herein we report for the first time the emergence of a pigmentation abnormality in the wire netting cod or longfin grouper (*Epinephelus quoyanus*) near Heron Island on the southern GBR. We began to encounter abnormally pigmented *E. quoyanus* from 2012 onwards as an incidental finding during unrelated research that required capture and tagging of *E. quoyanus* and stripey snapper (*Lutjanus carponotatus*) following on from research conducted in the same area in the mid-1990s (Diggles and Ernst 1997). Our awareness of the historical lack of pigment lesions in *E. quoyanus* and the recent emergence (since 2010) of superficially similar pigment lesions in another member of the Family Serranidae, namely the coral trout (*Plectropomus leopardus*), from the same region (Sweet *et al.* 2012; Lerebours *et al.* 2016) prompted us to publish these observations. Due to their presently unknown aetiology, we name these abnormalities serranid pigment abnormality syndrome (SPAS). This paper details the emergence of SPAS in the *E. quoyanus* population near Heron Island (Qld, Australia), describes the prevalence and gross pathology of the lesion and outlines the research that is needed to establish a better understanding of its geographical distribution, the course of disease and the mechanism(s) responsible for its emergence.

Materials and methods

The present study was conducted in the Scientific Research Zone and Conservation Park zones of the Great Barrier Reef Marine Park in the waters adjacent to Heron Island (23°15′54″S, 151°32′53″E) in the southern section of the GBR. Examinations of *E. quoyanus* ($n = 189$) were undertaken during five sampling trips between November 2010 and October 2017 (Table 1) and compared to historical data for *E. quoyanus* ($n = 158$) obtained in May 1996 during a previous study (Diggles and Ernst 1997). Fish were captured from both reef flat (water depth <2 m) and reef edge (water depths 2–10 m) environments using rod-and-reel methods and artificial lures with barbless hooks, as described previously (Diggles and Ernst 1997). The anatomical hooking location of each fish was noted, its total length (TL) was measured to the nearest millimetre and this was followed by examination of each fish for gross signs of SPAS lesions or other abnormalities. Each fish was then tagged with a plastic T-bar anchor tag with a unique identification number (TBA type; Hallprint, Hindmarsh Valley, SA, Australia) before being released at the site of capture. A smaller number of coral trout (*P. leopardus*; $n = 60$) were incidentally captured using similar methods in the same locations in 2016 and 2017 and were subjected to measurement and same examination before being released at the site of capture without tags. These activities were conducted under Great Barrier Reef Marine Park Authority permit numbers G08/24996.1, G011/34020.1, G13/35806.1 and G15/37589.1.

To determine whether SPAS-affected fish were significantly larger than unaffected fish, Student's two-tailed *t*-tests were conducted on fish length using online statistical programs (http://vassarstats.net/, accessed 20 March 2018).

Results

No pigment abnormalities were observed in any of the fish from the 1996, 2010 or 2011 studies, or in 42 of 43 fish examined in October and November 2012 (Fig. 1*a*). However, one fish (363 mm TL) captured on 1 November 2012 on the reef flat east of Heron Island was observed with conspicuous darkening and disorganisation of the brown 'wire netting' colouration pattern typical of this species over 100% of the head, 25–30% of the flanks and ~5% of the dorsal fin (Fig. 1*b*). To the naked eye, the lesions appeared smooth without obvious hyperplasia. Therefore, the sample prevalence for SPAS lesions in October and November 2012 was 1/43 = 2.33%, and, for all *E. quoyanus* captured since 1996 until that time, 1/218 = 0.46%.

In October 2016, an additional 64 *E. quoyanus* were examined, of which 4 (prevalence 6.25%) exhibited similar abnormally darkened pigmentation lesions without obvious hyperplasia most commonly involving the lower jaw, premaxilla and head (data not shown). Then, in October 2017, a further 65 *E. quoyanus* were examined, of which 11 (prevalence 16.92%) exhibited SPAS lesions most commonly on the lower jaw (prevalence 63.63%, mean coverage 52.14%), premaxilla (prevalence 36.36%, mean coverage 100%) and head (prevalence 72.72%, mean coverage 65.62%; Fig. 2), with involvement of the flanks (prevalence 45.45%, mean coverage 10–11%) and dorsal fin (prevalence 27.27%, mean coverage 6.67%; Fig. 3) in occasional fish (Table 2). After tagging and

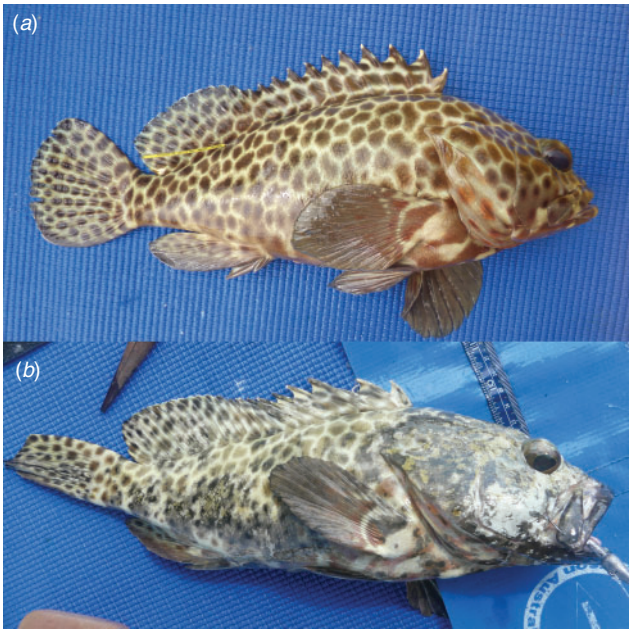

Fig. 1. (*a*) An *Epinephelus quoyanus* exhibiting the normal colouration pattern for this species. (*b*) The first *E. quoyanus* (363-mm total length) observed with serranid pigment abnormality syndrome (SPAS) captured at a depth of <2 m on the reef flat east of Heron Island on 1 November 2012. Note the conspicuous darkening and disorganisation of the brown 'wire netting' colouration pattern over the head and flank.

Fig. 2. An *Epinephelus quoyanus* (350-mm total length) captured from the reef flat south-east of Heron Island on 8 October 2017. Note the serranid pigment abnormality syndrome (SPAS) lesion over nearly the entire head, including 100% of the lower jaw and maxillae.

release, none of the SPAS-affected fish were subsequently recaptured during these sampling trips, hence information on whether the lesions observed are progressive or resolve over time is currently not available.

Fig. 3. Left flank and dorsal fin of an *Epinephelus quoyanus* (351-mm total length) captured from the reef flat south-east of Heron Island on 8 October 2017. Note the serranid pigment abnormality syndrome (SPAS) lesion over the flank and rear dorsal fin involving darkening and shrinkage of the natural honeycomb pattern.

Only *E. quoyanus* ≥325 mm TL captured on the reef flat exhibited SPAS lesions, with prevalence of lesions increasing markedly with fish size above this threshold (Table 3). Mean sample prevalence for pigmentation lesions for all *E. quoyanus* captured between November 2010 and October 2017 ($n = 189$) was $16/189 = 8.47\%$ (Table 3), being highest in October 2017, with an overall prevalence of 16.92% and a prevalence of 64.7% in fish >340 mm TL during that month (Table 1). The mean size of SPAS-affected *E. quoyanus* ($n = 16$; mean ± s.e.m. size 351 ± 12 mm TL, range 325–380 mm TL) was significantly greater than the mean size of unaffected fish ($n = 173$; mean ± s.e.m. size 293.8 ± 41.8 mm TL, range 145–370 mm; $P < 0.0001$, Students two-tailed *t*-test). It was not possible to weigh fish at the point of capture, but one of the SPAS-affected fish examined in October 2017 was recorded in field notes as being noticeably underweight compared with clinically normal fish of the same size (Table 2).

The only other species of fish around Heron Island observed with SPAS-like pigmentation lesions were coral trout (*P. leopardus*). In a sample of 21 *P. leopardus* captured in 2016, 2 were SPAS affected (overall prevalence 9.5%; Table 4), with both SPAS-affected fish being larger fish (>600 mm fork length (FL)) sampled from deeper waters (>10 m) from the channel between Heron and Wistari reefs ($n = 8$ fish sampled from the channel). In 2017, a sample of 39 *P. leopardus* captured from the channel, reef flat and reef edges near Heron Island revealed only 1 SPAS-affected fish (660 mm FL), which was caught and released from the reef edge (prevalence 2.56%; Fig. 4). The lesions on the coral trout were consistent in appearance with the melanised lesions reported by Sweet *et al.* (2012) to be present at a prevalence of 14.7% in the waters around Heron Island and nearby One Tree Island (Table 4).

Other than these two species, no abnormal pigmentation lesions were observed on any of the >1000 individual fish from >17 species captured and released by us during sampling trips

Table 2. Location and percentage coverage of serranid pigment abnormality syndrome (SPAS) lesions in individual *Epinephelus quoyanus* sampled from Heron Island in October 2017
TL, total length

Fish TL (mm)	Percentage coverage						Visually underweight
	Lower jaw	Premaxilla	Head	Left flank	Right flank	Dorsal fin	
360	100	100	75	5	5		No
380	100	100	100	15	15		Yes
350				10		10	No
360	100	100	70	10	10	5	No
351	5	100	90			5	No
340	10						No
355			100	10	20		No
355	20						No
350			10				No
342	30		70				No
353			10		5		No
Total number of affected individuals	7/11	4/11	8/11	5/11	5/11	3/11	1/11
Mean coverage (%)	52.14	100	65.62	10	11	6.67	–

Table 3. Prevalence of serranid pigment abnormality syndrome (SPAS) lesions in *Epinephelus quoyanus* examined at Heron Island between November 2010 and October 2017 versus fish total length
Only fish larger than 325 mm total length (TL) were affected, with the prevalence of SPAS increasing significantly with fish size

Fish TL (mm)	Number of normal fish	Number of fish with SPAS	Percentage of SPAS-affected fish in size class
0–150	2	0	0
151–200	2	0	0
201–250	17	0	0
251–300	72	0	0
301–320	34	0	0
321–350	35	8	18.60
>350	11	8	42.11
Number of all fish	173	16	8.46
Mean TL (mm)	293.8	351.0[A]	

[A] $P < 0.0001$ (Student's two-tailed *t*-test).

Table 4. Prevalence of serranid pigment abnormality syndrome (SPAS) lesions in coral trout (*Plectropomus leopardus*) sampled near Heron Island in October 2016 and 2017 compared with previously published data
Water temperature data are given as the mean ± range for October 2016 and October 2017, and as the range for the period 2010–12. FL, fork length

Year	Month	Water temperature (°C)	Number of fish sampled	FL range (mm)	SPAS prevalence (%)	Reference
2010–12	–	21–27	136	344–639	14.7	Sweet *et al.* (2012)
2016	October	24 ± 1	21	300–650	9.5	Present study
2017	October	25 ± 1	39	295–660	2.56	Present study

from 2010 onwards, including Lutjanidae (e.g. *Lutjanus carponotatus* ($n > 300$) and *Lutjanus bohar* ($n = 5$)), Lethrinidae (e.g. *Lethrinus nebulosus* ($n > 350$), *Lethrinus miniatus* ($n > 150$), *Lethrinus atkinsoni* ($n > 50$) and *Gymnocranius audleyi* ($n = 10$)), Labridae (e.g. *Cheilinus trilobatus* ($n > 20$), *Choerodon cyanodus* ($n > 20$) and *Choerodon venustus* ($n > 30$)), Carangidae (e.g. *Gnathanodon speciosus* ($n > 5$) and *Caranx melampygus* ($n = 1$)) and other species of Serranidae (*Epinephelus fasciatus* ($n > 50$), *Plectropomus maculatus* ($n > 5$), *Epinephelus fusgoguttatus* ($n = 2$), *Epinephelus cyanopodus* ($n = 2$), *Cephalopholis miniata* ($n = 5$) and *Variola louti* ($n = 3$)).

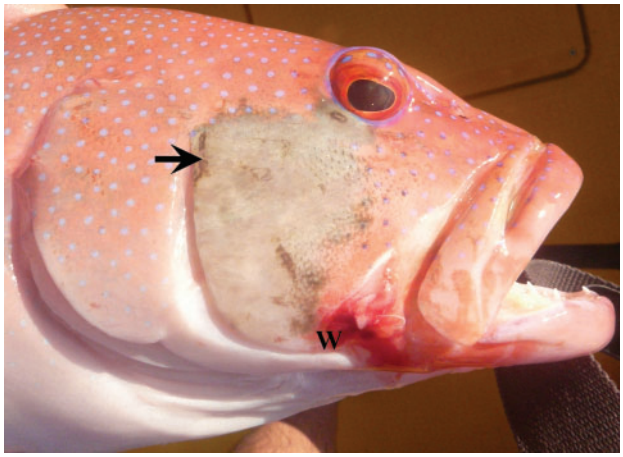

Fig. 4. An abnormal brown pigmented area (arrow) on the preopercule of a coral trout (*Plectropomus leopardus*) captured from the reef edge south-east of Heron Island. Similar pigmentation lesions were previously described from this species by Sweet *et al.* (2012). The haemorrhage in the mouth to the left of the maxilla is a hooking wound (W).

Discussion

Evidence from baseline studies conducted in the mid-1990s (Diggles and Ernst 1997), combined with observations of gross pathology from the fish examined here, confirm that the SPAS lesions in *E. quoyanus* at Heron Island are new, abnormal and may have emerged some time after 2010. No lesions were observed between 1996 and 2011, with the first lesion observed by us in 2012.

Abnormal skin pigmentation lesions involving melanisation were reported previously by Sweet *et al.* (2012) in specimens of another serranid, namely *P. leopardus*, sampled from near Heron and One Tree islands in the southern GBR since 2010. In that study, 20 fish of the 136 sampled were affected (prevalence 14.7%), with no apparent relationship between fish size and intensity of the pigmentation lesions, which appeared to have some characteristics of tumours composed of melanophore pigment cells (melanophoroma; see Sweet *et al.* 2012). We also observed superficially similar pigmentation lesions in *P. leopardus* (Fig. 4), but at a lower prevalence (9.5% in 2016, 2.5% in 2017). Because *P. leopardus* was not a target species for our studies, our capture methods and locations differed from those of Sweet *et al.* (2012) and, because we were not able to conduct pathological investigations to confirm lesion identity, our sample prevalences for *P. leopardus* for 2016 and 2017 are not directly comparable to their data from 2010 to 2012. Therefore, no conclusions should be drawn regarding possible changes in the prevalence of pigment lesions in *P. leopardus* over time based on our data.

We can confirm the pigmentation lesions we first observed in *E. quoyanus* in 2012 have increased in prevalence over time and show a strong positive relationship with fish size, with no fish <325 mm TL being affected and a high prevalence of lesions occurring in fish >340 mm TL (up to 64.7% in October 2017).

Sweet *et al.* (2012) reported that *P. leopardus* with skin lesions 'struck fishing hooks as strongly as healthy individuals, appeared to have good muscle tone and were assessed by external examination as healthy aside from the skin discolouration'. The three affected *P. leopardus* examined by us in the present study also appeared, by gross observation and behaviour, to be in normal condition. Similarly, most SPAS-affected *E. quoyanus* captured in the present study appeared to have normal body condition, although one fish was in notably poor condition (based on visual assessment). It should be noted that capture methods used in the present study (line fishing with lures) could bias prevalence data (and the possible capture of any clinically affected fish) if SPAS-affected fish were more or less likely than unaffected fish to strike lures. However, because the same sampling methods were used for our original study (Diggles and Ernst 1997), their use here is entirely appropriate because it allows direct comparison of recent data with the original baseline data from 1996. Any future studies could examine the use of alternative or supplementary methods for examining lesion prevalence, such as underwater visual census.

The conditions of our research permit (for a different study) did not allow for destructive sampling of *E. quoyanus* for histopathological or other biological analysis (e.g. aging and sexing fish), hence the pathology of the lesion and risk factors remain to be determined. However, based on their darkened appearance, it is possible that increased melanin is a characteristic of the lesions in *E. quoyanus*, as was described for *P. leopardus* by Sweet *et al.* (2012) and Lerebours *et al.* (2016). Of the various pigment cell tumours in fish, melanophoromas (melanoma) are common relative to other types of tumours (Singaravel *et al.* 2017). Malignant neoplastic melanomas may be aggressively invasive and hyperplastic (Okihiro 1988; Okihiro *et al.* 1992; Raloff 2012; Work and Aeby 2014) compared with benign melanomas or preneoplastic melanosis, the latter being the most common skin pigmentation lesion recorded in redfish (*Sebastes mentella*) from the North Atlantic (Bogovski and Bakai 1989). The lesions in *E. quoyanus*, like those of *P. leopardus*, do not protrude from the skin surface and hence do not appear to be significantly invasive, hyperplastic or malignant; however, as discussed previously, further pathological characterisation is necessary. In any case, the emergence of pigment lesions in both *P. leopardus* and *E. quoyanus* (but apparently not other fish species) from the same region of the GBR at approximately the same time (2010–12), warrants further investigation to establish causation and examine the course of disease.

Because the lesions in *E. quoyanus* have only recently emerged, nothing is known in relation to risk factors that may contribute to their causation, except for fish length (and presumably age). Bogovski and Bakai (1989) found that melanosis and melanophoromas in *S. mentella* were more common in certain regions of the North Atlantic, were positively correlated with fish size (only occurring in fish >26 cm, with prevalence exceeding 50% in fish >40 cm) and exhibited variation between sexes. In that study, pigmented lesions occurred in females two- to fivefold more often than in males, whereas in male fish 84% of pigment lesions occurred on the head and caudal fin (Bogovski and Bakai 1989). In *E. quoyanus* at Heron Island in October 2017, SPAS lesions were only observed on fish >340 mm TL, most commonly on the lower jaw (prevalence 63.63%) and head (prevalence 72.72%; Table 2). Exposure to

ultraviolet (UV) light was one of the potential risk factors discussed by Sweet *et al.* (2012) for pigmentation lesions in *P. leopardus*, and this could also be a risk factor for *E. quoyanus* given their shallow water (<2 m) habitat. In contrast, the melanised pigment lesions in *S. mentella* were unlikely to be related to UV exposure because those fish were taken in bottom or mid-water trawls at depths of 60–420 m, although the lesions were more common in fish taken at 'shallower' depths of 60–250 m (Bogovski and Bakai 1989).

Poor water quality is known to be associated with non-infectious diseases of fish, including neoplasms (Kinae *et al.* 1990). Recent water quality studies in Queensland have shown that biologically relevant reductions in water quality occur up to 100 km offshore during episodic flood events (Brodie *et al.* 2012). Heron Island is ~70 km offshore from mainland Queensland, and hence is within the range of exposure of flood plumes. However, Bogovski and Bakai (1989) sampled fish from the mid-Atlantic and suggested that melanised pigment lesions in *S. mentella* were not likely to be related to declines in water quality but due to aging-related factors (senescence) or reduced gene flow within certain fish populations. Reduced gene flow was also suggested by Sweet *et al.* (2012), although infectious aetiologies are also possible (Schmale *et al.* 2002; Work and Aeby 2014).

Fish length (and possibly age) is an important risk factor for SPAS in *E. quoyanus*, because lesion prevalence increases markedly once total fish length exceeds 325 mm. The maximum size of *E. quoyanus* is between 380 and 400 mm (Fishbase 2017), but the minimum legal size of *E. quoyanus* in Queensland is 380 mm. Although the fish population sampled at Heron Island during the present study exists in a research zone where the taking of fish without scientific permits is prohibited, this species is effectively protected throughout Queensland by fisheries regulations, and it is possible that similar size and age structures of *E. quoyanus* populations will occur outside marine park no-take areas closed to fishing. The occurrence of SPAS lesions in *E. quoyanus* populations beyond Heron Reef would require surveys designed to elucidate the geographic extent of this syndrome.

Given that SPAS lesions are more prevalent in larger fish, it is possible that these lesions are gross signs of senescence or a progressive disease. Further research is required to describe the course of disease in *E. quoyanus*, to establish causation and ascertain whether the condition is infectious. This could be done in wild fish by sampling affected fish from various locations to obtain pathological samples, genetic information and age at length and sex data. Tagging and recapturing of affected fish would allow temporal monitoring of the extent of the lesions to determine whether they resolve or progress and increase in severity or the area of affected skin over time. However, if natural mortality rates or tag loss rates are high, such an approach may not yield enough information, which would necessitate bringing affected fish into captivity to monitor whether the disease progresses over time under controlled conditions.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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